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10/523,723	11/29/2005	Toshihiro Tanaka	P26633	1079

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GREENBLUM & BERNSTEIN, P.L.C.
1950 ROLAND CLARKE PLACE
RESTON, VA 20191

EXAMINER	
SALMON, KATHERINE D	
ART UNIT	PAPER NUMBER
1634	

NOTIFICATION DATE	DELIVERY MODE
10/29/2008	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

gbpatent@gbpatent.com
pto@gbpatent.com

Office Action Summary	Application No.	Applicant(s)	
	10/523,723	TANAKA ET AL.	
	Examiner	Art Unit	
	KATHERINE SALMON	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *15 July 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *3-32* is/are pending in the application.

4a) Of the above claim(s) *4,10,11 and 13-32* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *3,5-9,12* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. This office action is in response to papers filed 7/15/2008.

2. Claims 3-32 are pending. Claims 1-2 have been cancelled.

3. Claims 4, 10-11 and 13-32 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 10/09/2007.

4. This application contains claims 4, 10-11 and 13-32 drawn to an invention nonelected with traverse in the reply filed on 10/09/2007. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01.

5. The following rejections for Claims 3, 5-9 and 12 are necessitated by amendment. Response to arguments follows.

6. This action is FINAL.

Withdrawn Objections and Rejections

7. The objection to the specification made in section 4 of the previous office action is moot based upon amendments to the claims and sequence listing. Specifically the sequence has been corrected to remove an extra nucleotide at the beginning of the sequence. Support for such a correction is found in Table 1 which specifically defines the claimed SNP mutation based upon amino acid sequence changes.

8. The rejections of the claims under 35 USC 112/2nd paragraph made in section 5 of the previous office action are moot, in part, based upon amendments to the claims and correction of the sequence listing.

9. The rejection of the claims under 35 USC 112/New Matter made in section 6 of the previous office action is moot based upon amendments to the claims and correction of the sequence listing.

10. The rejection of the claims under 35 USC 112/Written Description made in section 7 of the previous office action is moot based upon amendments to the claims and correction of the sequence listing.

11. The rejection of the claims under 35 USC 102(a) as anticipated by Ozaki et al. made in section 12 of the previous office action is moot based upon the 1.131 declaration filed 7/15/2008 which states that the inventors of the instant application are the sole inventors of the reference.

Claim Rejections - 35 USC § 112/2nd paragraph

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

12. Claims 3, 5-9, and 12 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 3 and 5-9 are indefinite because the claims do not recite a clear nexus between the preamble of the claims and the process steps of the claims. The preamble states a method for determining a vascular inflammatory disease. The method step is detecting a C/A polymorphism at nucleotide 80. Therefore the last step does not provide method steps for determining a vascular inflammatory disease; rather it provides a step for detecting a polymorphism. Therefore it is unclear if the claim is drawn to determining a vascular inflammatory disease or detecting a SNP.

Claim 12 is unclear over the preamble of "analyzing the expression state of LT-A". The method step of Claim 12 is detecting "a C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID No. 3". It is unclear how the detection step analyzes the expression state and as such it is unclear if the claim is drawn to analyzing the expression state or detecting a polymorphism.

Response to Arguments

The reply traverses the rejection. A summary of the arguments presented in the reply is provided below with response to arguments following.

The reply asserts that the claims have been amended to recite the presence of a polymorphism is indicative of the presence of an inflammatory disease (p. 11 3rd paragraph). This amendment has not been made to the claims, however.

This argument has been fully reviewed but has not been found persuasive.

The claims have not been amended in such a way to make it clear that the claims are drawn to determining vascular inflammatory disease or in the case of Claim

12 analyzing the expression state, because the claims still only are limited to positive active steps of detecting a polymorphism and do not require any steps of determining vascular inflammatory disease or analysis of expression state.

Claim Rejections - 35 USC § 112/New Matter

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

13. Claims 3, 5-9 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Claims 3, 5-9 are rejected as failing to comply with the written description requirement. Upon review of the specification, the specification does not appear to provide support for the recitation of “vascular inflammatory disease" in Claim 3. In response to the amendments, applicants have not pointed to any particular teaching in the specification.

The specification teaches inflammatory disease and provides examples of types of inflammatory disease including arterioscleroitc disease (p. 13 last paragraph), however, does not appear to provide support for a limitation of "vascular" types of inflammatory disease.

These amendments to the claims, therefore, constitute new matter.

Claim Rejections - 35 USC § 112/Scope of Enablement

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

14. Claims 3, 5-9, and 12 rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for determining myocardial infarction in a Japanese human which comprises detecting an AA in the nucleotide sequence of exon 3 of the LT-alpha gene by detection of the polymorphisms at nucleotide 80 in SEQ ID NO. 3, does not reasonably provide enablement for determining any vascular inflammatory disease in humans. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

Factors to be considered in determining whether a disclosure meets the enablement requirement of 35 USC 112, first paragraph, have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the

relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

Breadth of the claims

Claim 3 is drawn to a method for determining a vascular inflammatory disease comprising detecting SNP C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID no. 3 (referred to as C723A or T26N or Thr26Ala). Claim 5 is drawn to a method for determining a vascular inflammatory disease which comprises detecting a gene polymorphism whereby there is an amino acid change from threonine to asparagine. Claim 6 defines the disease as myocardial infarction. Claim 7 is defines the SNP position in Claim 3. Claims 8 and 9 define the method of detection and SNP position in Claim 3. Claim 12 is drawn to a method for analyzing the expression state of LT-α gene by detection of SNP C723A.

The claims are broadly drawn to determining any vascular inflammatory disease by detection of SNP C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID no. 3.

When the claims are read in light of the specification, the specification does not provide predictable guidance for any vascular inflammatory disease by detection of SNP C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID no. 3. The art, as presented below, that such correlations are unpredictable and population specific.

Nature of the Invention

The claims are broadly drawn to determining any vascular inflammatory disease

by detection of SNP C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID no. 3.

The invention is in a class of invention which the CAFC has characterized as "the unpredictable arts such as chemistry and biology." Mycogen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Fed. Cir. 2001).

Teachings in the Specification and state of the art

The specification discloses that LT-α gene is one of the cytokines produced during the earliest phase of the process of angiitis and it activates the cytokine cascade by inducing other mediators (p. 2 2nd paragraph). The specification discloses that these mediators are known to be involved in atheroma formation and aterma lesions (p. 2 2nd paragraph).

The specification discloses a method for determining inflammatory diseases involving identifying gene polymorphism associated with the disease (p. 3 2nd paragraph). The specification discloses that the invention has typed SNPs within a population of about 1000 myocardial infarction patients and a control group of about 1000 persons by multiplex PCR-invader assay (p. 3 3rd paragraph). However, the specification has not provided a predictable correlation of a specific SNP with any vascular inflammatory disease.

The specification discloses that the C/A polymorphism at nucleotide 80 of the nucleotides sequence of exon 3 of the LT-α gene shown in SEQ ID No. 3 causes an amino acid mutation from threonine to asparagine in codon 26 (p. 13 1st full paragraph).

However, this mutational change has not been correlated with any vascular inflammatory disease. The specification asserts a correlation between the specific SNP and myocardial infarction. However, the art, as presented below teaches that correlation between a specific type of inflammatory disease and the specific SNP cannot be extrapolated to any vascular inflammatory disease (see Witte et al.). Post-filing art further teaches that a correlation of the amino acid change represented by this SNP is not correlated to myocardial infarction (Tobin et al.). Therefore though, the specification provides some statistical correlation between a particular SNP and a specific inflammatory disease, the post-filing correlation that this correlation is unpredictable.

The specification asserts that inflammatory disease is not specifically limited, as long as it is a disease confirmed to induce cell adhesion factors or cytokines that are know to correlate with pathologic conditions of inflammation (p. 13 last paragraph).

The specification provides examples of inflammatory disease include chronic articular inflammation, rheumatism, lupus, inflammatory enteritis, allergic reactions, bacterial shock, and myocardial infarction (p. 13 last paragraph).

The term “vascular inflammatory disease” is not specifically defined and encompasses a myriad of diseases which would include any inflammatory disease that has an effect of the vascular system, each with different genetic associations. It would be unpredictable for one of skill in the art to extrapolate a correlation between a specific disease and a SNP to any vascular inflammatory disease. It would be unpredictable because each disease is genetically different and it is unpredictable that a change in

one amino acid sequence would be able to detect any of these diseases because it is unclear if the amino acid sequence change is associated to any disease. The art teaches that extrapolation to any disease is unpredictable. As discussed below Witte et al. that there was no statistically significant correlation between NcoI LT-A mutation and asthma (e.g. a vascular disease).

The specification asserts that the expression level is significantly higher where codon 26 encodes threonine versus encoding asparagine (p. 13 1st full paragraph). The specification in figure 5 discloses that TNF-inducing activity and Selectin E0inducing activity in human coronary-artery endotheilial cells express mRNA coding for threonine significantly higher than mRNA encoding for asparagine. However, Claim 12 is drawn to analyzing any expression state of the gene by detection of the SNP the claim is not drawn to detection of changes in expression between the two mRNAs which code for different amino acid sequences and wherein one expression level is higher.

In summary, the claims are drawn to determining any vascular inflammatory disease. The specification however only discloses the correlation of one vascular inflammatory disease, myocardial infarction. The art, as discussed below, teaches that extrapolation of one correlation between a specific disease and a specific SNP to other diseases is unpredictable. Further the post filing art teaches that the correlation of myocardial infarction and the codon change T26N (threonine to asparagine) is not predictable associated (Tobin et al.).

Therefore the skilled artisan would have to perform a large amount or experimentation in order to correlate the particular in the LT-A gene to any vascular

inflammatory disease. There would be many intervening steps the skilled artisan would have to perform without any guarantee of success to practice the invention as claimed.

Working Examples

The specification provides a Japanese population in which 1133 patients have been diagnosed with myocardial infarction (p. 23 last paragraph. P. 24 1st paragraph). The specification asserts that SNP were detected using the invader PCR assay method (p. 24).

The specification asserts that 94 myocardial infarction patients were genotypes and the allelic frequency was compared to a population of healthy subjections (p. 26 1st paragraph). 26 SNPs were types and expanded by sample size (p. 27 1st paragraph). Table 1 indicates a sample size of 1133 myocardial infarction patients and 1006 control patients. The specification asserts that the population was genotyped (table 1).

Therefore the specification teaches that of the 26 SNPs detected on 1133 myocardial infarction patients only 3 SNPs in LT-A had a statistically significant association. For SNP C723A there are three possible genotypes (CC, CA, and AA) (e.g. the claimed SNP) (Table 1). The specification discloses that there is a predictable correlation of homozygous (AA) individuals with myocardial infarction compared to homozygous wild type (CC) and heterozygous (CA). Therefore the p-value discloses in Table 1 is based on the detection of the "A" allele on both strands of nucleic acid. The claims, however, are encompass detecting one "A" allele. Therefore it is unpredictable

that there is a correlation of the allelic mutation based on the correlation of the homozygous AA in the population.

The predictability or unpredictability of the art and degree of experimentation

Post-filing art teaches that SNPs in LT-A are not strongly associated with any vascular inflammatory diseases. Witte et al. (European Journal of Human Genetics 2002 Vol 10 p 82) teaches detection of the LT-A SNP of the first intron NcoI recognition sequence in asthma and nonasthmatic patients (abstract). Witte et al. teaches no statistically significant correlation between this SNP and asthma (p. 84 1st paragraph). Therefore Witte et al. teaches that LT-A mutations are not associative to any inflammatory disease.

The art teaches that associations between SNPs in LT-A and vascular inflammatory disease are unpredictable. Trabetti et al. (Journal Med Genet 1999 VOl. 36 p. 323) teaches an association of atopy in asthma patients and the LT-A NocI SNP (abstract). However, Trabetti et al. teaches this same association in other populations (Busselton population) was not observable (p. 324 last paragraph and p. 325 1st paragraph). Therefore Trabetti et al. teaches that in different populations the association of the LT-A mutation with a specific disease in not correlative.

The art teaches that SNP associations to myocardial infarction are unpredictable. Newton-Cheh et a. (JAMA 2004 Vol. 291 p. 3008) teaches that myocardial infarction is a complex trait to which multiple environmental and genetic factors contribute (p. 3008 2nd paragraph). Newton-Cheh et al. teaches that there is also evidence that there are

sex differences between male and females with regard to correlation of genetic variants in myocardial infarction (p. 3008 3rd paragraph). Therefore the art teaches the unpredictability of such associations in different populations.

Newton-Cheh et al. teaches that the studies which are published have conflicting results because of population differences (p. 3008 last 2 paragraphs). Newton-Cheh et al. teaches that results are often confounded by baseline covariates (p. 3009 1st column 2nd full paragraph), correlation of genetic variants with other variants that are casual (p. 3009 2nd column 2nd paragraph) and false positive and negatives given the number of SNPs and population sizes (p. 3009 last two paragraphs).

Post-filing art discloses that there is no predictive correlation between myocardial infarction and the elected SNP (e.g. Thr26Asn). Tobin et al. (European Heart Journal 2004 Vol 25 p. 459) teaches a method of detecting SNP from LT-A and the association with myocardial infarction (abstract). Tobin et al. tested 1052 subjects in Caucasian population (abstract). Tobin et al. does not teach a statistically significant p-value for SNP thr26asn (p= 0.446) (Table 2 p. 462 last gene and polymorphism). Tobin et al. teaches that though MI was associated with Thr26Asn in other populations it was not in his study (p. 465 1st column last paragraph). Tobin et al. teaches that the difference in association might be due to the different types of populations (p. 465 2nd column 1st paragraph). Therefore even the statistically significant association of the SNP with myocardial infarction in the instant specification (Table 1 SNP Thr26Asn to myocardial infarction) is not reproducible in other populations.

Amount of Direction or Guidance Provided by the Specification

The specification does not provide any specific guidance as to how to correlate determination of any vascular inflammatory disease with detection of the specific SNP in the LT-A gene.

The specification discloses a method of detecting many SNPs from LT-A and correlating the mutations to one specific inflammatory disease (myocardial infarction). The specification teaches that only 3 of the SNPs tested were statistically correlated to myocardial infarction.

The art teaches that associations between mutations of LT-A gene and vascular inflammatory disease are unpredictable and population specific. The art teaches that even the mutation T26N (the elected SNP) is not correlative to myocardial infarction in post filling art.

Quantity of Experimentation

The quantity of experimentation in this area is extremely large since there is significant number of parameters, which would have to be studied prior to being able to practice the claimed invention as broadly as written.

The skilled artisan would have to analyze the association of the elected SNP to determine its association with any vascular inflammatory disease. However, neither the specification nor the art provides guidance as to extrapolate a correlation of a specific disease to any inflammatory disease. The skilled artisan would then need to test associations in a variety of populations because the art teaches that associations are population specific.

This would require significant inventive effort, with each of the many intervening steps, upon effective reduction to practice, not providing any guarantee of success in

the succeeding steps.

Level of Skill in the Art

The level of skill in the art is deemed to be high.

Conclusion

Case law has established that '(t)o be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without 'undue experimentation.'" *In re Wright* 990 F.2d 1557, 1561. *In re Fisher,* 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) it was determined that '(t)he scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art". The amount of guidance needed to enable the invention is related to the amount of knowledge in the art as well as the predictability in the art. Furthermore, the Court in *Genetech Inc. v Novo Nordisk* 42 USPQ2d 1001 held that '(I)t is the specification, not the knowledge of one skilled in the art that must supply the novel aspects of the invention in order to constitute adequate enablement".

In the instant case, the specification does not provide a predictable correlation of detection of the SNP C/A polymorphism at nucleotide 80 in the nucleotide sequence of exon 3 as shown in SEQ ID No. 3 to vascular inflammatory disease. Further, the art teaches that such correlations are unpredictable and population specific (see Tobin et al).

Accordingly, in view of the unpredictability in the art, and the lack of disclosure in

the specification and in the prior art and the unpredictability of the art, it would require undue experimentation for one of skill in the art to make and use the claimed invention.

Response to Arguments

The reply traverses the rejection. A summary of the arguments presented in the reply is provided below with response to arguments following.

The reply asserts that the claims have been amended to overcome the enablement rejection (p. 12 3rd full paragraph).

This argument has been fully reviewed but has not been found persuasive.

Though the claims have been limited to a particular SNP and vascular inflammatory disease, as discussed above, the art teaches that such associations are unpredictable. Specifically, Tobin et al. teaches that the elected SNP is not associated with myocardial infarction. This indicates that there is unpredictability in the art with regard to the association of the specific SNP and vascular inflammatory disease. However, the response does not present any arguments as to how the instant specification overcomes such unpredictability in the association.

Conclusion

15. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP

§ 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

16. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Katherine Salmon whose telephone number is (571) 272-3316. The examiner can normally be reached on Monday-Friday 8AM-430PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Katherine Salmon
Examiner
Art Unit 1634

/Juliet C Switzer/

Primary Examiner, Art Unit 1634